



THE UNIVERSITY *of* EDINBURGH

Edinburgh Research Explorer

“Most People are Simply Not Designed to Eat Pasta”: Evolutionary Explanations for Obesity in the Low-Carbohydrate Diet Movement

Citation for published version:

Knight, C 2011, "Most People are Simply Not Designed to Eat Pasta": Evolutionary Explanations for Obesity in the Low-Carbohydrate Diet Movement', *Public Understanding of Science*, vol. 20, no. 5, pp. 706-719. <https://doi.org/10.1177/0963662510391733>

Digital Object Identifier (DOI):

[10.1177/0963662510391733](https://doi.org/10.1177/0963662510391733)

Link:

[Link to publication record in Edinburgh Research Explorer](#)

Document Version:

Peer reviewed version

Published In:

Public Understanding of Science

Publisher Rights Statement:

© Knight, C. (2011). "Most People are Simply Not Designed to Eat Pasta": Evolutionary Explanations for Obesity in the Low-Carbohydrate Diet Movement. *Public Understanding of Science*, 20(5), 706-719.
[10.1177/0963662510391733](https://doi.org/10.1177/0963662510391733)

General rights

Copyright for the publications made accessible via the Edinburgh Research Explorer is retained by the author(s) and / or other copyright owners and it is a condition of accessing these publications that users recognise and abide by the legal requirements associated with these rights.

Take down policy

The University of Edinburgh has made every reasonable effort to ensure that Edinburgh Research Explorer content complies with UK legislation. If you believe that the public display of this file breaches copyright please contact openaccess@ed.ac.uk providing details, and we will remove access to the work immediately and investigate your claim.



“Most people are simply not designed to eat pasta”:¹ Evolutionary explanations for obesity in the low-carbohydrate diet movement

Christine Knight

ESRC Genomics Policy and Research Forum, University of Edinburgh

ESRC Genomics Forum
College of Humanities and Social Science
University of Edinburgh
St John's Land, Holyrood Road
Edinburgh
EH8 8AQ
Scotland UK
Tel: +44 131 651 4747
Fax: +44 131 651 4748
Email: christine.knight@ed.ac.uk

Low-carbohydrate diets, notably the Atkins Diet, were particularly popular in Britain and North America in the late 1990s and early 2000s. Based on a discourse analysis of bestselling low-carbohydrate diet books, I examine and critique genetic and evolutionary explanations for obesity and diabetes as they feature in the low-carbohydrate literature. Low-carbohydrate diet books present two distinct neo-Darwinian explanations of health and body-weight. First, *evolutionary nutrition* is based on the premise that the human body has adapted to function best on the diet eaten in the Paleolithic era. Second, the *thrifty gene theory* suggests that feast-or-famine conditions during human evolutionary development naturally selected for people who could store excess energy as body fat for later use. However, the historical narratives and scientific arguments presented in the low-carbohydrate literature are beset with generalisations, inconsistencies and errors. These result, I argue, from the use of the primitive as a discursive “blank slate” onto which to project ideals perceived to be lacking in contemporary industrialised life.

Keywords: diet; genetics; obesity; diabetes; primitivism

Low-carbohydrate diets, notably the Atkins Diet, were particularly popular in the late 1990s and early 2000s in Britain, the United States and other English-speaking Western nations.

Recommending the reduction or elimination of starchy and sugary foods, and a focus on non-starchy vegetables, meat and fish, they include the notorious Atkins Diet, as well as the South Beach Diet, the Zone, Sugar Busters and Protein Power (Atkins, 2002; Agatston, 2003; Sears, 1995; Steward et al., 1998; Eades and Eades, 1996). In this article I approach the recent low-carbohydrate trend as one response to the twin obesity and diabetes epidemics. Based on a discourse analysis of bestselling low-carbohydrate diet books, I trace the deployment of two distinct neo-Darwinian

explanations of health and body-weight in the low-carbohydrate diet movement. From each of these two explanations – first, evolutionary nutrition, and second, the thrifty gene theory – arises a corresponding set of recommendations for today's dieters.

Relative to the thrifty gene theory, evolutionary nutrition has not been well explored by science and technology studies, and one of the main goals of this article is to provide a detailed critique of its incarnation in the low-carbohydrate diet literature. The thrifty gene theory, on the other hand, and the geneticisation of obesity and diabetes in general, have been considered in somewhat more detail by previous social scientific and other critiques (Fee, 2006; Gard and Wright, 2005; McDermott, 1998; Paradies, Montoya and Fullerton, 2007; Poudrier, 2003, 2007). While I draw on this body of work in this article, my purpose in discussing the thrifty gene theory here is not to rehearse that critique in detail, but to examine the inflection of the thrifty gene theory in popular nutrition writing, specifically the hugely popular low-carbohydrate diet literature. Further, my ultimate goal in this paper is to consider the relationship – and tension – between evolutionary nutrition and the thrifty gene theory as they appear in low-carbohydrate diet books: the two models are seemingly complementary, but in fact sit uneasily within the same paradigm.

Evolutionary nutrition is a relatively straightforward concept, based simply on the premise that the human body has adapted to function best on the diet eaten in the Paleolithic era. The thrifty gene theory (or thrifty gene hypothesis) is somewhat more complex. It suggests that feast-or-famine conditions during human evolutionary development naturally selected for people whose bodies were efficient in their use of food calories (those who could store excess energy as body fat for later use). Unfortunately, in contemporary conditions of constant dietary abundance, the so-called “thrifty gene” predisposes people to diabetes and obesity. This dual investment in human evolutionary origins and genetic design as the guiding principles of proper nutrition mandates close

attention to Paleolithic or “primitive” diet, which therefore functions as the ultimate blueprint for contemporary low-carbohydrate regimes (Knight, 2005, 2006, 2008).

Both evolutionary nutrition and the thrifty gene theory are the subject of intense research and debate outside the context of low-carbohydrate dieting. Each model has its own scholarly and popular literature, its own high-profile advocates, and its own set of critics. Evolutionary nutrition as a specific field of study is generally agreed to have begun with the publication of Eaton and Konner's article “Paleolithic Nutrition” in the *New England Journal of Medicine* (1985). Eaton, Konner and Shostak followed this with the popular diet book *The Paleolithic Prescription* (1988). Today, the field of evolutionary nutrition is arguably spearheaded by Loren Cordain, Professor of Health and Exercise Science at Colorado State University. Cordain is the author of the popular *Paleo Diet* (2001) as well as numerous peer-reviewed scientific articles.² He has coauthored articles with other popular diet book writers, including Michael and Mary Dan Eades, authors of *Protein Power* (Cordain, Eades and Eades, 2003). At their most extreme, evolutionary nutritionists advocate a strict “Paleo” diet, excluding virtually all post-agricultural foods. Their maxim is “could I eat this if I were naked with a sharp stick on the savanna?” (Audette, 1999). However, evolutionary nutrition also encompasses less extreme low-carbohydrate regimes which take Stone-Age diet as a flexible prototype, either for the types of foods that dieters should eat, or the appropriate balance of macronutrients (protein, carbohydrate and fat).

The other neo-Darwinian model to appear in the popular low-carbohydrate literature, the thrifty gene theory, was originally proposed by geneticist and human biologist James Neel (1962). Neel revised and expanded his thrifty gene hypothesis in two further publications (1982, 1999) before his death in 2000. The thrifty gene theory has been taken up enthusiastically in scientific and popular explanations for both diabetes and obesity, especially in relation to Indigenous groups

amongst whom the prevalence of these disorders is disproportionately high. As Paradies, Montoya and Fullerton write,

the racialized incarnation of [the thrifty gene] hypothesis continues to outlive its progenitor[,] as it continues to be reiterated and researched in relation to Indigenous Australians, Native Americans, and First Nation Canadians. (2007: 210)

The racialised version of the thrifty gene theory has recently prompted its own small but rich multidisciplinary critical literature, of which the work of Paradies, Montoya and Fullerton forms a part. Other contributors to this critique include McDermott (1998), Poudrier (2003, 2007) and Fee (2006).

1. Evolutionary nutrition and *The Zone*

Dr. Atkins' New Diet Revolution, *Sugar Busters*, *The Zone* and *Protein Power* all draw on the evolutionary nutrition model, often in combination with the thrifty gene theory. However, compared with other low-carbohydrate diet books, *The Zone* (Sears, 1995) posits an extremely early date in the history of life on earth as the end-point of human evolution. Sears treats human evolutionary adaptation as being “essentially” complete well before the Paleolithic era; in fact, well before the emergence of humankind. He argues that “by the time man came along” the hormonal “control systems” (such as insulin) were already “deeply embedded in his genes” (100). In Sears's logic, Paleolithic diet therefore functions as a kind of “test case” for human health. According to this line of reasoning, archeological evidence which indicates the state of human health in Paleolithic times can tell us whether or not Paleolithic diet was “in sync” with humankind's well-established biochemistry. Drawing on the work of Eaton and others (Eaton and Konner, 1985; Eaton, 1992; Eaton, Shostak and Konner, 1988), Sears asserts that “in Neo-Paleolithic times both men and women had the bone structures of world-class athletes” (1995: 101). Moreover, he claims, Paleolithic people grew to a comparable height to people in affluent countries today: “The average height of Neo-Paleolithic man was about five feet ten, and for Neo-Paleolithic women about five

feet six” (103). The apparently exceptional health and fitness of Stone-Age men and women confirms for Sears that the Paleolithic “menu” of “lean meat, fruits, and vegetables” was “in harmony with human genetic makeup” (101). He attributes the health effects of this diet above all to its macronutrient balance, and the effect of this on hormonal regulation of blood sugar:

almost to the percentage point *Neo-Paleolithic diets had the same protein-to-carbohydrate ratio as a Zone-favorable diet*. So that Neo-Paleolithic diet kept insulin, glucagon, and eicosanoid responses on an even keel. (101, original italics)

Sears concludes that since human genes have not changed “substantially” since well before the Paleolithic era, dieters today can achieve the exceptional health and fitness of our ancestors by mimicking Stone-Age nutrition.

The claim that the macronutrient ratio of a “Zone-favorable diet” matches that of Neo-Paleolithic diets “almost to the percentage point” raises a number of issues. Sears attributes this claim directly to Eaton and Konner (1985), who suggest that human diets in the late Paleolithic period averaged 34 percent protein and 45 percent carbohydrate, with the remaining 21 percent coming from fat (288). The ratio of protein-to-carbohydrate in this estimate (0.75) certainly does match the ideal ratio proposed by Sears elsewhere in *The Zone* (65), although the percentage figures are somewhat different. The Zone Diet is defined by its macronutrient ratio: 30 percent protein, 40 percent carbohydrate and 30 percent fat. However, Eaton and Konner acknowledge that their figures are only averages: early human beings, they suggest, probably derived anywhere between 20 and 50 percent of their diet from animal foods, with the remainder from plant foods (1985: 285).³ When animal foods make up 20 percent of the diet, 24.5 percent of energy comes from protein and 55 percent from carbohydrate, a ratio of about 0.45 (Eaton and Konner, 1985: 287). This is well outside Sears’s “Zone-favorable” range of between 0.6 and 1 (Sears, 1995: 65). In order to make Paleolithic diets fit his own model of optimum nutrition, Sears simply discounts differences in diet between geographic regions and historical periods within the Neo-Paleolithic era. He also assumes that macronutrient intakes were the same for men, women, children and adults, and ignores the

possibility of seasonal dietary variation. In their critical examination of obesity discourse, Gard and Wright (2005) review research on energy intake and expenditure in prehistoric populations. Based on their appraisal of the literature, they question whether it will ever be possible to estimate the energy intake of prehistoric people with any degree of accuracy, given the length of time that has elapsed between then and now (Gard and Wright, 2005: 111). By extension, I would argue that our knowledge of the *macronutrient* breakdown of prehistoric diets is equally “only ever likely to be extremely imprecise” (Gard and Wright, 2005: 111), belying the numerical exactitude Sears claims.

A further problem with Sears's approach lies in his assertion that macronutrient ratio may be taken as the defining feature of diet. In their concluding comparison of Paleolithic and modern American diets, Eaton and Konner note that as well as eating more protein than modern Americans, Paleolithic people consumed much more fibre, calcium, iron, folate and vitamins. They also ate much less sodium than modern Americans, and much less total fat. Further, “the fat they ate was substantially different from ours [...] the paleolithic diet had [...] more essential fatty acids, and a much higher ratio of polyunsaturated to saturated fats” (Eaton and Konner, 1985: 288). Sears himself points out that Neo-Paleolithic diets were “exceptionally rich in micronutrients,” before passing quickly on to macronutrient composition (101). It is certainly arguable that high micronutrient intake or some other distinguishing feature of the Paleolithic diet was what made Stone-Age people so healthy (assuming, for the moment, that they were). Sears acknowledges that “man needs a *modern version* of a Neo-Paleolithic diet,” effectively conceding that we cannot replicate Paleolithic diets exactly (103, *italics added*). Not only is our knowledge of prehistoric eating habits inevitably hazy, but the massive social and ecological changes that have occurred since the Stone Age have irrevocably changed the foods available to us. It is not clear why Sears privileges macronutrient ratio as the defining feature of healthy diet – a pragmatic possibility is that

it is relatively easy for today's dieters to mimic. At any rate, Sears uses the Neo-Paleolithic data selectively in what appears to be a post-hoc rationalisation of his macronutrient paradigm.

Having established that Paleolithic diet was ideal because it matched human genetic inheritance, Sears sets out to show that subsequent human diets have diverged from that inheritance, especially in the introduction of grains and dairy products. Making the widely-recognised claim that the majority of the world's adult population is lactose-intolerant, Sears argues that this is due to a lack of evolutionary adaptation to dairy consumption:

Only with the domestication of cattle some eight thousand years ago did cow's milk [...] become widely available. The only populations which eventually evolved to retain the activity of the lactase enzyme in adulthood were those who were constantly exposed to lactose through relentless consumption of dairy products – primarily Europeans of Scandinavian descent. As a result, these people can still digest lactose as adolescents and adults. / Unfortunately, 80 percent of the world's population has not yet caught up to the Scandinavians. [...] Maybe with another twenty thousand years of evolution, every human will be able to digest dairy products, but that's certainly not the case now. (102)

The genetic basis of lactase persistence is generally accepted in the scientific literature, although the theory that lactase persistence is an evolutionary adaptation to pastoralism remains a hypothesis, and would seem to be inherently unprovable (Swallow, 2003: 213; Vesa, Marteau and Korpela, 2000: 166S). Further, although Sears does not say so, a growing body of research suggests that lactose tolerance is not *solely* genetically heritable, but may be built up in “lactose maldigesters” via regular exposure to dairy foods (Hertzler and Savaiano, 1996; Pribila et al., 2000; Vesa, Marteau and Korpela, 2000). Sears's evolutionary argument and vocabulary reflect a hierarchical model of evolutionary development in which the rest of the world's population is “behind” northern Europeans. Curiously, despite his awareness of lactose intolerance and his recognition that dairy products were not part of the Stone-Age diet, Sears recommends a wide variety of both fermented and non-fermented dairy products as part of the Zone regime, an inconsistency he shares with Atkins.

Crucially, Sears's evolutionary explanation for lactose tolerance directly contradicts his insistence that human evolutionary adaptation was complete well before the Paleolithic era, as discussed earlier. In his discussion of lactose tolerance, Sears concedes that genetic adaptations have occurred in a significant minority of the human population in response to relatively recent changes in diet. He also makes a similar concession in relation to grain-foods. Initially, Sears tries to emphasise the “sluggishness” of human adaptation to grains over the 10,000 years that have elapsed since the agricultural revolution:

Remember that from an evolutionary point of view ten thousand years is nothing more than the flick of an eyelash. Genomes – a species' total genetic makeup – don't change much in ten thousand years. So human genes have been adapting very reluctantly and very sluggishly to the introduction of these two new food groups [dairy foods and grains] ten thousand years ago. In fact, by and large *humankind has been genetically unable to cope with these foods*. (102, original italics)

However, Sears has already argued earlier in *The Zone* that around one-quarter of the American population *is* genetically equipped to eat large quantities of carbohydrate without ill effect (30, 65). He is therefore forced to hypothesise that a significant minority of Americans *have* adapted to the recent introduction of grain-foods into the human diet:

Just as constant exposure to dairy products has allowed most northern Europeans to evolve genetically [to be] able to tolerate milk, I suspect that constant exposure to grain has begun to create a slow evolutionary adaptation toward reducing the typically elevated insulin response to high-density carbohydrates [...]. Maybe in twenty thousand years, all humans will be able to eat high-density carbohydrates without an exaggerated insulin response. (103)

To summarise then, Sears admits that around 20 percent of the world's people have adapted to tolerate dairy foods, and around 25 percent of Americans have adapted to tolerate grain products, over the mere “flick of an eyelash” in evolutionary time. In Sears's own terms, this achievement is not “sluggish” at all. These concessions are by no means negligible. They undermine Sears's entire dietary prescription, which is based on the premise that human evolutionary adaptation somehow ceased millions of years ago, and that we must therefore attend to our prehistoric biochemistry to determine the optimum diet for people today. If, on the other hand, a substantial minority of the world's population has continued to adapt over those millions of years, and has even adapted

effectively to dietary changes over much shorter periods of time, it seems unlikely that the rest of the world's population has not adapted at all, perhaps in as-yet-unlooked-for and unknown ways. Ethnobiologist Gary Paul Nabhan (2004) critiques the “Paleolithic prescription” on the basis that different ethnoracial groups today arguably display microevolutionary adaptations to their recent historical environments which have vital consequences for nutrition and health (36-62). An important example is thalassemia, which confers resistance to malaria (Nabhan, 2004: 63-91).⁴ But even if the rest of humanity has somehow stayed the same genetically over the last 10,000 years, the recent adaptations that a substantial minority has undergone should surely influence how *this* group, at least, should eat (presumably by consuming more grains, dairy products, or both). *The Zone* is thus not particularly helpful in telling *me* what *I* should eat, except to imply that I should perhaps identify my own “genetic code” through either genetic testing or dietary experimentation, both of which would seem to render *The Zone*'s regime redundant.

2. Evolutionary nutrition and *Dr. Atkins' New Diet Revolution*

In part, the logical inconsistencies in Sears's version of evolutionary nutrition stem from his penchant for numerical precision. *The Zone* confidently demarcates clear-cut historical periods, finely-balanced macronutrient ratios, and precise population fractions. By contrast, *Dr. Atkins' New Diet Revolution* (Atkins, 2002) presents a relatively hazy and romanticised evolutionary account, which nonetheless differs subtly in its details from that of *The Zone*. As I noted above, Sears posits an extremely early end-point to the history of human evolutionary adaptation, arguing that human evolution was essentially complete well before the Paleolithic era. By contrast, Atkins treats the Stone Age as the “evolutionary window” during which human nutritional adaptation took place. In other words, Atkins suggests that human evolutionary processes abruptly stopped at the end of the

Paleolithic era. Atkins and Sears thus agree that a Paleolithic-style low-carbohydrate diet is the eating pattern to which humans are best adapted, but their reasoning is different. Rather than constituting a “test case,” Stone-Age diet is significant to Atkins's logic because it *produced* the human body as it is today:

the human body evolved and primitive humans thrived as hunter-gatherers who subsisted primarily on meat, fish, vegetables, fruit, whole grains and seeds and nuts. Candy bars were few and far between. The human body is used to dealing with unrefined foods as they occur in Nature. Consequently, your body's capacity to deal with an excess of processed foods is pretty poor, which is why our twenty-first-century way of eating so often gets us into trouble. (48)

Unlike Sears, who highlights the macronutrient composition of Paleolithic diet (as I have discussed), Atkins focuses here and elsewhere on the *types* of foods that “primitive” people ate: “meat, fish, vegetables, fruit, whole grains and seeds and nuts.” Of course, whole grains were a rare feature of Stone-Age diets, and were never consumed in large quantity. Nonetheless, the defining absence in this list is refined carbohydrate of any kind. Atkins reasons that if these are the foods on which humanity evolved, then today's dieters should also “thrive” on this diet.

Atkins's evolutionary account poses many of the same difficulties that I discussed above in relation to *The Zone*. In particular, Atkins describes evolution as an *event* in the past tense, rather than an ongoing *process*. As noted above, this approach neglects the possible import of more recent evolutionary change. Atkins's representation of evolution as a finite occurrence also leads, inevitably, to a concept of evolution as homogeneous. Evolution in Atkins's construction is a historical event which happened everywhere and for everyone at the same time and in the same way, producing a human body that does not vary across either time or space. In the passage cited above, the phrase “the human body” (singular, homogeneous) appears twice. In its third iteration this body becomes “*your* body,” which is apparently exactly the same as all other human bodies in its (in)capacity to deal with processed and refined foods. In one sense, this theory of human homogeneity might be applauded, since it avoids the tendency we witnessed in *The Zone* to treat certain ethnoracial groups as evolutionarily “backward.” But the drawback of this model is that it

cannot recognise that rates of obesity and related diseases (such as type 2 diabetes and cardiovascular disease) differ markedly in different parts of the world and also within individual nations. I am certainly not advocating more nuanced evolutionary explanations for global health disparities. Rather, I suggest that evolutionary nutrition is an inherently unsatisfactory model no matter how carefully it is deployed. At its most simplistic, evolutionary nutrition obscures stark inequalities in health within and between nations. Where evolutionary nutritionists do acknowledge recent adaptive change, they risk replicating racist hierarchies of evolutionary development. But even in its most acceptable forms, evolutionary nutrition remains inherently deterministic and preservationist, denying self-determination and individual agency in favour of strict genetic prescription.

In low-carbohydrate discourse healthy diet is defined in opposition to a monolithic modern Western diet, the distinguishing feature of which is large quantities of highly refined carbohydrates. The ostensible “robustness” of Stone-Age man functions in this discursive system as an expression of discontent with the overweight and diseased bodies that modern Western foodways have arguably caused. The “radical relativity of meaning” (Bell, 1972: 4) that marks modern representations of the primitive means that the logic of evolutionary nutrition is inevitably circular. Representations of that which is *not* modern and *not* Western in low-carbohydrate discourse thus tend to be highly idealised and generalised in order to fit the binaristic model (Knight 2008). The representations of Stone-Age diet, health and lifestyle that I have discussed in this section are consistently contradictory and unsupported by scientific and historical evidence. It is striking, for example, that Sears's claims about the macronutrient composition of Paleolithic diet are not substantiated even in the specific paper that he himself cites in support of his figures. Instead, authors like Sears and Atkins betray a discursive pressure to make the foodways of other times and other places fit their own preconceived notions of healthy diet.

3. The thrifty gene hypothesis in the low-carbohydrate diet literature

The insistence on the evolutionary origins of obesity and diabetes in texts such as *The Zone* and *Atkins* paves the way for the interpolation of the thrifty gene theory, which again appears in the majority of bestselling low-carbohydrate diet books. I begin here by quoting the description of the purported thrifty gene mechanism in *The South Beach Diet* (Agatston, 2003), as it is particularly clearly stated, though without naming the thrifty gene as such:

We've been genetically conditioned to store fat since the dawn of *homo sapiens*, as a survival strategy to see us through times of famine. / The problem now, of course, is that we never experience the famine end of that equation, only the feast. (9)

Later Agatston explains:

Our bodies are designed to store excess energy (which we call calories) for a very good reason: For most of humanity's existence, securing a steady and sufficient supply of food has been our biggest, most important challenge. Feast or famine prevailed and, to adapt, our bodies would save the energy from today's feast, knowing that tomorrow it [*sic*] would need to burn saved fuel in order to survive. That's why this particular brand of obesity concentrates the fat in the mid-section – it leaves the extremities lithe and muscular, for ease of manual labour and, especially, flight. Advanced civilization has done a great deal to eradicate famine, but at the expense of our waistlines and our cardiovascular systems, which now suffer from the fact that we store fat we no longer need. (70)

As these passages from *South Beach* make clear, the thrifty gene hypothesis theorises obesity and diabetes as the result of a mismatch between an evolutionary genotype favouring energy efficiency and fat storage, and the constant abundance of the modern diet. I noted earlier that a racialised version of the thrifty gene theory is often employed today to explain the particularly high rates of diabetes and obesity amongst many Indigenous groups. However, Neel's original hypothesis (1962) was that the thrifty gene might confer a predisposition to diabetes and obesity on individuals from any ethnoracial background, including Europeans. In his final publication on the thrifty gene (1999), Neel explicitly rejected a simple genetic explanation for high rates of diabetes amongst Native Americans (S2-3). The two passages from *South Beach* cited above reflect Neel's broader, species-wide version of the hypothesis. Indeed, *South Beach* perhaps expands the ambit of

the thrifty gene theory even further, seeming to suggest that *all* modern humans possess a thrifty genetic capacity to accumulate body fat. As I noted earlier, this homogeneous model of evolutionary adaptation avoids the racist trap of blaming obesity and diabetes amongst Indigenous people on their supposed evolutionary backwardness. But the species-wide version of the thrifty gene theory also fails to explain why not everyone in the world, even in affluent Western nations, is obese, thus masking the socioeconomic, environmental and lifestyle factors which might be responsible for disparities in health and weight. As Gard and Wright point out, “What is needed here are clear and specific arguments about concrete events that have caused increasing overweight and obesity in some, but not all, communities” (2005: 111).

It is important to stress that Neel only ever proposed the thrifty gene theory as a hypothesis. Indeed, the theory would seem to be inherently “unfalsifiable” (Gard and Wright, 2005: 111), since “a feature of adaptive scenarios such as the TGH [thrifty gene hypothesis] is that they cannot be explicitly tested” (Paradies, Montoya and Fullerton, 2007: 217). Yet scholarly and popular publications on obesity and diabetes – including low-carbohydrate diet books – routinely present the thrifty gene theory as fact. Poudrier (2003) comments aptly that “[a]lthough its existence has not been confirmed scientifically, the ‘thrifty gene’ theory often appears as an assumed truth seemingly waiting (almost impatiently) for scientific authorization” (127). Critics also question the historical feast-or-famine assumption that underpins the thrifty gene theory: the belief that prehistoric hunter-gatherer life was necessarily characterised by alternating conditions of “feast” or famine, which would make the capacity to store body-fat advantageous (Poudrier, 2007: 247). Certain hunter-gatherer groups, including Pacific Islander peoples who now suffer extremely high rates of diabetes and obesity, historically “were free of feast-and-famine cycles altogether” (Paradies, Montoya and Fullerton, 2007: 212).

Protein Power (Eades and Eades, 1996) provides a prime example of this slippage between hypothesis and fact in its narration of the thrifty gene theory. Like Agatston in *South Beach*, Eades and Eades take for granted that famine was an inevitable part of early human existence:

There has been discussion in the scientific community for years about the so-called “thrifty gene.” First used with reference to diabetes, this phrase has come to mean the genetic material that has been passed along to us by our prehistoric ancestors that allows us to better survive hunger and privation. Since periodic famines, brought on by game scarcity, heavy winters, droughts, or other natural disasters, were a part of prehistoric life, it makes sense that the people best suited to these deprivations would live to reproduce. Obviously this happened. Natural selection culled the weak and left a population that had the biochemistry and physiology necessary to squeeze every possible calorie from the food at hand and store it efficiently. (405)

The passage opens cautiously, referring to “discussion” about the thrifty gene, and flagging its hypothetical status with the modifier “so-called.” Initially, the authors alternate between present, past and conditional tenses. By arguing that the thrifty gene theory is a logical thesis, they implicitly acknowledge its speculative nature: “*it makes sense* that the people best suited to these deprivations *would live* to reproduce” (italics added). But the tense abruptly switches and the thrifty gene hypothesis becomes fact: “Obviously this happened”! The passage concludes confidently in the simple past tense.

The corollary to the assumption that famine was an inevitable part of pre-agricultural life is the blithe assertion that human nutrition today is uniformly characterised by “feast” conditions. For instance, in the passages cited earlier from *South Beach*, we have a species-wide ambit, implying that all the world's people now share an abundant and secure supply of food. Such blanket explanations for obesity and diabetes gloss over the continued existence of famine in the third world, as well as high rates of food insecurity and malnutrition amongst poor and marginalised groups within the West. Further, in Western countries, those groups most likely to experience food insecurity are also those with the highest rates of obesity and diabetes. That the wealthiest and most privileged people in Western societies are *not* the most overweight belies Agatston's claim that the “problem” lies simply with over-abundance and over-availability of food. *South Beach* certainly does not suggest

that dieters should starve themselves periodically in order to be thin and healthy. Yet it states quite clearly that our “problem” is that we no longer experience famine (9, cited above).

South Beach is unusual amongst the low-carbohydrate diet books I examine in this article in presenting a conventional caloric version of the thrifty gene theory, in which stored body-fat equates simply to surplus calories consumed. Other low-carbohydrate diet texts modify the thrifty gene hypothesis to reflect the arguably differential effects of dietary macronutrients (protein, carbohydrate and fat). The “heresy” of many popular low-carbohydrate diets is that they reject the conventional caloric paradigm, holding instead that dietary carbohydrate mediates weight gain and loss (BBC Horizon, 2004). The biological mechanism by which diet authors graft carbohydrate onto the thrifty gene theory is that of insulin response, as in the following passage from *The Zone* (Sears, 1995):

Insulin responses evolved to cope with the uncertainty of the food supply under extreme, potentially faminelike conditions. If animals or humans are forced to go long periods between meals (as is often the case when food comes from hunting or gathering), then the ability to store nutrients can make the difference between life and death. / When times are leaner – between meals, for example, or during fasts – declining insulin levels mean a corresponding increase in levels of glucagon. This, in turn, tells the liver to release stored carbohydrates in a controlled, measured way so as to keep the brain fed and maintain adequate mental function. / [... T]he release of stored body fat is your safety net during famine. Just as a runner could potentially finish twenty marathons using only stored body fat as fuel, you could live for about forty days without eating, on your stored body fat alone. (100)

Notice the difference between *The Zone*'s argument and that from *South Beach*. *The Zone* refers to “stored *carbohydrates*” and the “ability to store *nutrients*,” whereas *South Beach* refers to the body's capacity to “store excess *energy*.” The passage above does not specifically explain why the evolutionary insulin response might cause people today to gain weight, but previous passages in the book make this connection, explaining that “insulin is essentially a storage hormone, evolved to put aside *excess carbohydrate calories* in the form of fat in case of future famine. So the insulin that's stimulated by *excess carbohydrates* aggressively promotes the accumulation of body fat” (15, italics added).

Atkins explains the human capacity to store and then “burn” body-fat in similar evolutionary terms. Like Sears, Eades and Eades, Atkins identifies insulin as the hormonal regulator of these processes:

Before the invention of agriculture, in the first few hundred thousand years of human life, periods of severe food shortage must have been uncomfortably common. Human beings had to be able to burn their own body fat for fuel on those recurrent occasions when the larder was bare. Naturally, our bodies devised a highly efficient system for doing just that. / Have you ever wondered what sustained bears and other hibernating animals during their long winter sleep? It was the utilization of their fat stores. When you dial down the volume of insulin production, as you do in lipolysis, your body is equipped to burn your own body fat in a similar way.⁵ (60)

Importantly, there is a fundamental contradiction between this version of the thrifty gene theory and Atkins's model of evolutionary nutrition, discussed earlier in this article. In evolutionary nutrition, Paleolithic diet is held up as the optimal diet for people today. This evolutionary logic depends on the premise that Paleolithic people were exceptionally fit, lean and healthy; Paleolithic health is taken as proof that the Stone-Age diet was, and continues to be, the optimal evolutionary eating pattern for human physical functioning. By contrast, the thrifty gene theory presupposes subsistence conditions in the Paleolithic era, marked by alternating periods of feast and famine, weight gain and loss. In the low-carbohydrate diet literature, evolutionary nutrition and the thrifty gene theory thus collide, appropriately, over the question of carbohydrate consumption and excess weight in Paleolithic times. Evolutionary nutrition holds that Stone-Age people ate consistently low levels of unrefined carbohydrate, and no refined carbohydrate at all. According to Atkins and authors like him, this is what made Paleolithic diet so healthy. Indeed, the omission of refined carbohydrate and the restriction of unrefined carbohydrate are treated as the defining features of Stone-Age nutrition, and the pattern that dieters should emulate today. On the other hand, the thrifty gene theory (as stated in *The Zone*, *Protein Power* and *Atkins*) maintains that Paleolithic people stored excess carbohydrate as body-fat; this was the crucial evolutionary adaptation which allowed them to survive times of famine. But the key question here is: *what* excess carbohydrate? In the absence of high-carbohydrate grains or concentrated sugars in the Stone-Age diet, low-carbohydrate logic

suggests that Paleolithic people would simply never have stored body fat. This negates any purported survival advantage in times of famine which might naturally select for the “thrifty gene.”

Low-carbohydrate diets, obesity and type 2 diabetes

Evolutionary nutrition and the thrifty gene theory are both fundamental to the low-carbohydrate literature's investment in primitive health and diet. Both of these evolutionary models maintain that the answer to the question “what should we eat” can only be found by turning to the primitive past. Low-carbohydrate authors therefore follow the logical path mapped out by more radical evolutionary nutritionists such as Eaton and Cordain. In this article, I have shown how low-carbohydrate texts theorise obesity and diabetes as the inevitable result of a mismatch between the Stone-Age body and modern Western eating habits. In my discussion of *The Zone*, *Dr. Atkins' New Diet Revolution*, *The South Beach Diet* and *Protein Power* I have repeatedly identified generalisations, unsubstantiated assertions and inconsistencies, including between the two explanatory models themselves in this context. These logical and evidential difficulties result, I suggest, from the use of the primitive as a blank slate onto which to project ideals perceived to be lacking in contemporary Western life.

Given these difficulties, we need to consider alternative evidence for the efficacy and safety of low-carbohydrate diets. Clinical trials prompted by the popular low-carbohydrate diet trend quickly established that low-carbohydrate diets are effective for weight-loss in the short term. Indeed, up to 6 months, study participants lose more weight on a low-carbohydrate diet than a “traditional” low-fat and/or low-calorie diet, while at 12 months the two remain at least equally effective (Nordmann et al., 2006; Hession et al., 2009). In addition, low-carbohydrate diets have been shown to improve glucose control in type 2 diabetes (Kirk et al., 2008). Although critics

expected negative effects on markers of cardiovascular disease risk, in fact low-carbohydrate plans improve dieters' levels of triglycerides and "good" HDL cholesterol, though evidence for their effects on LDL ("bad") cholesterol remains equivocal (Hession et al., 2009: 46). Researchers have expressed concern that "individual data indicate a large degree of variability in the magnitude and [...] direction" of change in LDL levels (Volek, Sharman and Forsythe, 2005: 1339), suggesting (as I argued earlier in this paper) that no single dietary plan may be right for everyone.

Studies of weight-loss dieting outside the clinical trial setting indicate that a low-carbohydrate diet can be a successful choice long-term for at least some individuals (Feinman, Vernon and Westman, 2006; Phelan et al., 2007). However, scientific evidence for the long-term effects of low-carbohydrate dieting remains scarce, although randomised controlled trials lasting up to 3 years have been published (Cardillo, Seshadri and Iqbal, 2006), and further long-term research is in progress. Recent large observational cohort studies over 20 years of follow-up suggest that lower dietary carbohydrate levels are not in themselves associated with an increased risk of either coronary heart disease or death, but that the *types* of protein and fat in a low-carbohydrate diet make a difference. A low-carbohydrate diet emphasising animal sources of protein and fat was associated with higher all-cause mortality, while a low-carbohydrate diet based on vegetable sources carried a decreased risk of overall mortality, and of death from cardiovascular disease in particular (Halton et al., 2006; Fung et al., 2010). As argued elsewhere in this paper, these studies suggest that macronutrient breakdown is only part of the wider nutritional picture.

The popularity of low-carbohydrate diets in the 1990s and 2000s was prompted, at least in part, by concern about rising rates of obesity and type 2 diabetes, and the perceived failure of low-fat/low-calorie dietary advice to address these "epidemics". As I have stressed throughout this paper, both obesity and type 2 diabetes are strongly associated not only with poor nutrition and

physical inactivity but with poverty. Yet individualised diet regimes – including low-carbohydrate diets – take no account of socioeconomic or health inequalities, whether within nations or globally. Not only have low-carbohydrate diets been criticised for their expense (Raffensperger, 2008), but the blanket prescription of an “evolutionarily appropriate” diet obscures inequalities in health and nutrition within and between nations, challenging public health efforts aimed at addressing differences in health and body weight between different population groups. In this context, community-based nutrition interventions are required to address the social and environmental causes of unhealthy eating habits, beginning in childhood, as well as the lifestyle factors associated with obesity and related health conditions.⁶ Even more fundamentally, reducing current rates of obesity and type 2 diabetes will require policies to address broader socioeconomic inequality, known to be associated with overweight, ill-health and disease.

This research was carried out as part of a PhD project jointly supported by the University of Adelaide and Australia's Commonwealth Scientific and Industrial Research Organisation (CSIRO). This paper was revised as Policy Research Fellow at the ESRC Genomics Policy and Research Forum, University of Edinburgh. The support of both CSIRO Human Nutrition and the Economic and Social Research Council (ESRC) is gratefully acknowledged. I would like to thank Angus Clarke, Isabel Fletcher, Lisa Moran and my PhD supervisors Heather Kerr and Carlene Wilson for their feedback at various points.

¹ The quotation in the article title is from *The Zone* (Sears, 1995: 204).

² A full list of papers is available at www.thepaleodiet.com/published_research/index.shtml.

³ Eaton and Konner derive this percentage range from a review of the diets of contemporary hunter-gatherer groups who live ‘in an inland, semitropical habitat’ similar to that of Paleolithic hunter-gatherers. I have critiqued the identification of contemporary hunter-gatherers with Paleolithic people elsewhere (Knight, 2005).

⁴ Although Nabhan's research offers a helpful counterpoint to low-carbohydrate discourse, I have serious concerns about the implications of his work, which is highly prescriptive and deterministic. Nabhan acknowledges the ‘problem’ of hybrid ethnoracial identities – the fact that following one's ancestral diet is deceptively difficult in countries where one's ethnicity is likely to be a ‘hodgepodge’ (Heldke, 2003: 161). However, *Why Some Like It Hot* fails to provide any practical nutritional advice to the many people who are ‘mutts rather than blue bloods’ (38). Although Nabhan recognises that race is a social category, not a genetic one (51–54), *Why Some Like It Hot* proceeds on the basis of racial genetic homogeneity, arguing that particular ethnoracial groups should behave in specified ways because of their genetic makeup. I am sympathetic to many of the community-based nutrition interventions Nabhan describes, but would prefer them to be judged on their contribution to individual and community health, rather than the blanket imperative to ‘reconnect’ with ancestral foodways.

⁵ Atkins uses the term lipolysis here to refer to ketosis, the metabolic state induced when the diet is very low in carbohydrate and the body switches to using fat for energy.

⁶ See, for example, the recent special issue of the journal *Obesity* (February 2010, volume 18, issue 1S) on community-based interventions for childhood obesity.

- Agatston, A. (2003) *The South Beach Diet: The Delicious, Doctor-Designed Plan for Fast and Healthy Weight Loss*. London: Headline.
- Atkins, R. C. (2002) *Dr. Atkins' New Diet Revolution*. New York: HarperCollins.
- Audette, R. (1999) *Neanderthin*. New York: St Martin's Press.
- BBC Horizon (2004) "The Atkins Diet," URL (consulted 29 July 2010): <http://www.bbc.co.uk/science/horizon/2004/atkinstrans.shtml>
- Bell, M. (1972) *Primitivism*. London: Methuen.
- Cardillo, S., Seshadri, P. and Iqbal, N. (2006) "The Effects of a Low-Carbohydrate versus Low-Fat Diet on Adipocytokines in Severely Obese Adults," *European Review for Medical and Pharmacological Sciences* 10: 99-106.
- Cordain, L. (2001) *The Paleo Diet: Lose Weight and Get Healthy by Eating the Food You Were Designed to Eat*. Hoboken, NJ: John Wiley & Sons.
- Cordain, L., Eades, M. R. and Eades, M. D. (2003) "Hyperinsulinemic Diseases of Civilization: More Than Just Syndrome X," *Comp Biochemistry and Physiology* 136: A95-112.
- Eades, M. R. and Eades, M. D. (1996) *Protein Power*. New York: Bantam.
- Eaton, S. B. (1992) "Humans, Lipids and Evolution," *Lipids* 27: 814-20.
- Eaton, S. B. and Konner, M. (1985) "Paleolithic Nutrition: A Consideration of its Nature and Current Implications," *New England Journal of Medicine* 312(5):283-9.
- Eaton, S. B., Shostak, M. and Konner, M. (1988) *The Paleolithic Prescription*. New York: Harper & Row.
- Fee, M. (2006) "Racializing Narratives: Obesity, Diabetes and the 'Aboriginal' Thrifty Genotype," *Social Science & Medicine* 62: 2988-97.
- Feinman, R. D., Vernon, M. C. and Westman, E. C. (2006) "Low Carbohydrate Diets in Family Practice: What Can We Learn from an Internet-Based Support Group," *Nutrition Journal* 5: 26.
- Fung, T. T., Van Dam, R. M., Hankinson, S. E., Stampfer, M., Willett, W. C. and Hu, F. B. (2010) "Low-Carbohydrate Diets and All-Cause and Cause-Specific Mortality," *Annals of Internal Medicine* 153: 289-98.
- Gard, M. and Wright, J. (2005) *The Obesity Epidemic: Science, Morality and Ideology*. London: Routledge.
- Halton, T. L., Willett, W. C., Liu, S., Manson, J. E., Albert, C. M., Rexrode, K. and Hu, F. B. (2006) "Low-Carbohydrate-Diet Score and the Risk of Coronary Heart Disease in Women," *New England Journal of Medicine* 355: 1991-2002.
- Heldke, L. M. (2003) *Exotic Appetites: Ruminations of a Food Adventurer*. New York & London: Routledge.
- Hertzler, S. R. and Savaiano, D. A. (1996) "Colonic Adaptation to Daily Lactose Feeding in Lactose Maldigesters Reduces Lactose Intolerance," *American Journal of Clinical Nutrition* 64(2): 232-6.
- Hession, M., Rolland, C., Kulkarni, U., Wise, A. and Broom, J. (2009) "Systematic Review of Randomized Controlled Trials of Low-Carbohydrate vs. Low-Fat/Low-Calorie Diets in the Management of Obesity and its Comorbidities," *Obesity Reviews* 10: 36-50.
- Kirk, J. K., Graves, D. E., Craven, T. E., Lipkin, E. W., Austin, M. and Margolis, K. L. (2008) "Restricted-Carbohydrate Diets in Patients with Type 2 Diabetes: A Meta-Analysis," *Journal of the American Dietetic Association* 108: 91-100.
- Knight, C. (2005) "'The Food Nature Intended You to Eat': Low-Carbohydrate Diets and Primitivist Philosophy," in L. Heldke, K. Mommer and C. Pineo (eds) *The Atkins Diet and*

- Philosophy: Chewing the Fat with Kant and Nietzsche*, pp. 43-56. Chicago and La Salle, Illinois: Open Court.
- Knight, C. (2006) "Nostalgia and Authenticity in Low-Carbohydrate Dieting," in R. Hosking (ed.) *Authenticity in the Kitchen: Proceedings of the Oxford Symposium on Food and Cookery 2005*. Totnes, Devon: Prospect Books.
- Knight, C. (2008) "'The Food Nature Intended You to Eat': Nutritional Primitivism in Low-Carbohydrate Diet Discourse. PhD diss., University of Adelaide, South Australia.
- McDermott, R. (1998) "Ethics, Epidemiology and the Thrifty Gene: Biological Determinism as a Health Hazard," *Social Science & Medicine* 47(9): 1189-95.
- Nabhan, G. P. (2004) *Why Some Like It Hot: Food, Genes, and Cultural Diversity*. Washington: Island Press.
- Neel, J. V. (1962) "Diabetes Mellitus: A 'Thrifty' Genotype Rendered Detrimental by 'Progress'?" *American Journal of Human Genetics* 14: 353-62.
- Neel, J. V. (1982) "The Thrifty Genotype Revisited," in J. Koberling (ed.) *The Genetics of Diabetes Mellitus*. London: Academic Press.
- Neel, J. V. (1999) "The 'Thrifty Genotype' in 1998," *Nutrition Reviews* 57(5): S2-9.
- Nordmann, A. J., Nordmann, A., Briel, M., Keller, U., Yancy, W. S., Jr, Brehm, B. J. and Bucher, H. C. (2006) "Effects of Low-Carbohydrate vs Low-Fat Diets on Weight Loss and Cardiovascular Risk Factors," *Archives of Internal Medicine* 166: 285-93.
- Paradies, Y. C., Montoya, M. J. and Fullerton, S. M. (2007) "Racialized Genetics and the Study of Complex Diseases: The Thrifty Genotype Revisited," *Perspectives in Biology and Medicine* 50(2): 203-27.
- Phelan, S., Wyatt, H., Nassery, S., Dibello, J., Fava, J. L., Hill, J. O. and Wing, R. R. (2007) "Three-Year Weight Change in Successful Weight Losers who Lost Weight on a Low-Carbohydrate Diet," *Obesity* 15(10): 2470-7.
- Poudrier, J. (2003) "'Racial' Categories and Health Risks: Epidemiological Surveillance among Canadian First Nations," in D. Lyon (ed.) *Surveillance as Social Sorting: Privacy, Risk, and Digital Discrimination*, pp. 111-34. London and New York: Routledge.
- Poudrier, J. (2007) "The Geneticization of Aboriginal Diabetes and Obesity: Adding Another Scene to the Story of the Thrifty Gene," *Canadian Review of Sociology and Anthropology* 44(2): 237-61.
- Pribila, B. A., Hertzler, S. R., Martin, B. R., Weaver, C. M. and Savaiano, D. A. (2000) "Improved Lactose Digestion and Intolerance among African-American Adolescent Girls Fed a Dairy-Rich Diet," *Journal of the American Dietetic Association* 100(5): 524-8.
- Raffensperger, J. F. (2008) "The Least-Cost Low-Carbohydrate Diet is Expensive," *Nutrition Research* 28(1): 6-12.
- Sears, B. (1995) *The Zone: A Dietary Road Map*. New York: HarperCollins.
- Steward, H. L., Bethea, M. C., Andrews, S. S. and Balart, L. A. (1998) *Sugar Busters! Cut Sugar to Trim Fat*. New York: Ballantine.
- Swallow, D. M. (2003) "Genetics of Lactase Persistence and Lactose Intolerance," *Annual Reviews in Genetics* 37: 197-219.
- Vesa, T. H., Marteau, P. and Korpela, R. (2000) "Lactose Intolerance," *Journal of the American College of Nutrition* 19(2): 165-75S.
- Volek, J. S., Sharman, M. J. and Forsythe, C. E. (2005) "Modification of Lipoproteins by Very Low-Carbohydrate Diets," *Journal of Nutrition* 135(6): 1339-42.

This is the author's final manuscript version of a piece published in *Public Understanding of Science*, 20:5 (2011), pp. 706-719. Please refer to the published version for citation, as copy-editing and other changes will have been made. If you have difficulty accessing the published version, email christine.knight@ed.ac.uk.

Dr Christine Knight completed her PhD on primitivism in the low-carbohydrate diet movement at the University of Adelaide, South Australia, in 2008. The project was jointly supported by the Commonwealth Scientific and Industrial Research Organisation (CSIRO). Since then she has been employed as Policy Research Fellow at the ESRC Genomics Policy and Research Forum, University of Edinburgh, where she has a remit in knowledge exchange and public engagement. Her research interests lie at the intersection of literary and cultural studies, food studies, and science and technology studies.